Modern Concepts of Cardiovascular Disease

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DR. SAMUEL A. LEVINE, Boston, *Editor*
DR. MARSHALL N. FULTON, Boston, *Associate Editor*

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THE MECHANISM OF HEART FAILURE

PART II

Nocturnal dyspnea is one of the most distressing features of heart failure. It may be manifest in three ways. (1) Increasing dyspnea during the latter part of the day, reaching its height when the patient tries to sleep, and undoubtedly is due to increasing pulmonary engorgement. Several hours complete rest before attempting to sleep often gives great relief. (2) Severe attacks of periodic or Cheyne-Stokes breathing often disturb the early hours of sleep. This is particularly so in cases of failure due to hypertension or arteriosclerosis. In addition to pulmonary engorgement there are cerebral vascular changes which impair the local circulation, which appear to aggravate the oxygen want to the respiratory centre, leading to distressing paroxysmal hyperpnea followed by apnea with complete relief for a short period, only to have a recurrence of respiratory distress, which periodicity may be repeated for a considerable time. Continuous inhalation of oxygen and a small dose of morphia give great relief. (3) Acute paroxysms of dyspnea when in deep sleep may give the patient most alarming distress. They are associated with, if not caused by, acute edema of the lungs. Many moist râles are present and frothy sputum may be abundant. The upright position and the inhalation of oxygen until the pulmonary edema has subsided gives temporary relief. The fear and restlessness is lessened by morphia.

Cyanosis in heart failure is principally due to interference with respiratory function which is the direct result of pulmonary congestion or engorgement. It is recognized by the presence of fine râles indicating a transudate of serum into the alveoli or bronchioles, although a certain degree of engorgement may be present without recognizable transudation. Reference has already been made to pulmonary engorgement reducing respiratory function. In the first instance alveolar ventilation may be reduced to such an extent as to allow the partial pressure of oxygen in the alveoli to fall to a level where the venous blood is not normally oxygenated. This will naturally become effective on exertion and has been referred to as one of the causes of dyspnea. At first cyanosis (blue tinge to the skin and mucous membranes), due to abnormal amounts of reduced hemoglobin in the arterial blood will be apparent on severe exertion, but as heart failure progresses it will appear on less and less effort until it will be present when at rest. At some point another factor in addition to reduced alveolar ventilation appears, namely, direct interference with the diffusion of oxygen through the alveolar walls on account of swelling of the alveolar cells and transudate into the lumen of the alveoli and bronchioles. As heart failure progresses still farther, the transudate fills the bronchi and even the trachea with a copious serous fluid which may or may not be blood tinged. In this state the cyanosis is usually extreme and requires energetic treatment. We are here really dealing with edema of the lungs which varies in degree from time to time and may progress insidiously or develop with explosive suddenness, practically suffocating the patient with abundant frothy pinkish serous fluid which may coagulate to a solid mass on boiling. If the arterial blood be examined for its oxygen saturation this will be found to be reduced from a slight degree to almost desaturation and the cyanosis will be in direct proportion.

There are instances when cyanosis may be present but the arterial blood normally saturated. In these persons it is presumed that this is due to the slow flow of blood through the peripheral capillaries leading to an abnormal increase of reduced hemoglobin in mixed capillary blood. It is usually patchy in distribution and the color is purplish. It also appears as if the capillaries were engorged; in fact, in these areas an increased concentration of the erythrocytes is usually to be detected.

A ready means of differentiating cyanosis due to interference with aeration of the blood from that due to capillary stagnation is the following: in the former the cyanosis will be removed or greatly reduced by the inhalation of air enriched with oxygen, while if due to the latter the submersion of a cyanosed part, such as the hand, in hot water will usually change the purplish color to a bright red.

The degree of arterial anoxemia sufficient to

produce definite cyanosis is a menace to the organism as a whole. However, this is less so when it develops over a prolonged period of time than when it suddenly occurs, as the organism cannot accommodate itself with sufficient rapidity to meet the dire consequences resulting from such a vital upset in its internal environment.

Edema of heart failure is quite characteristic appearing, as it usually does, in the feet in the evening only to be gone in the morning, and then gradually progressing in the duration and extent until the whole body, including the lower arms, is waterlogged up to the level of the heart. As would be expected from its progress, the edema is most marked in the dependent parts, and in the looser tissues such as the genitals.

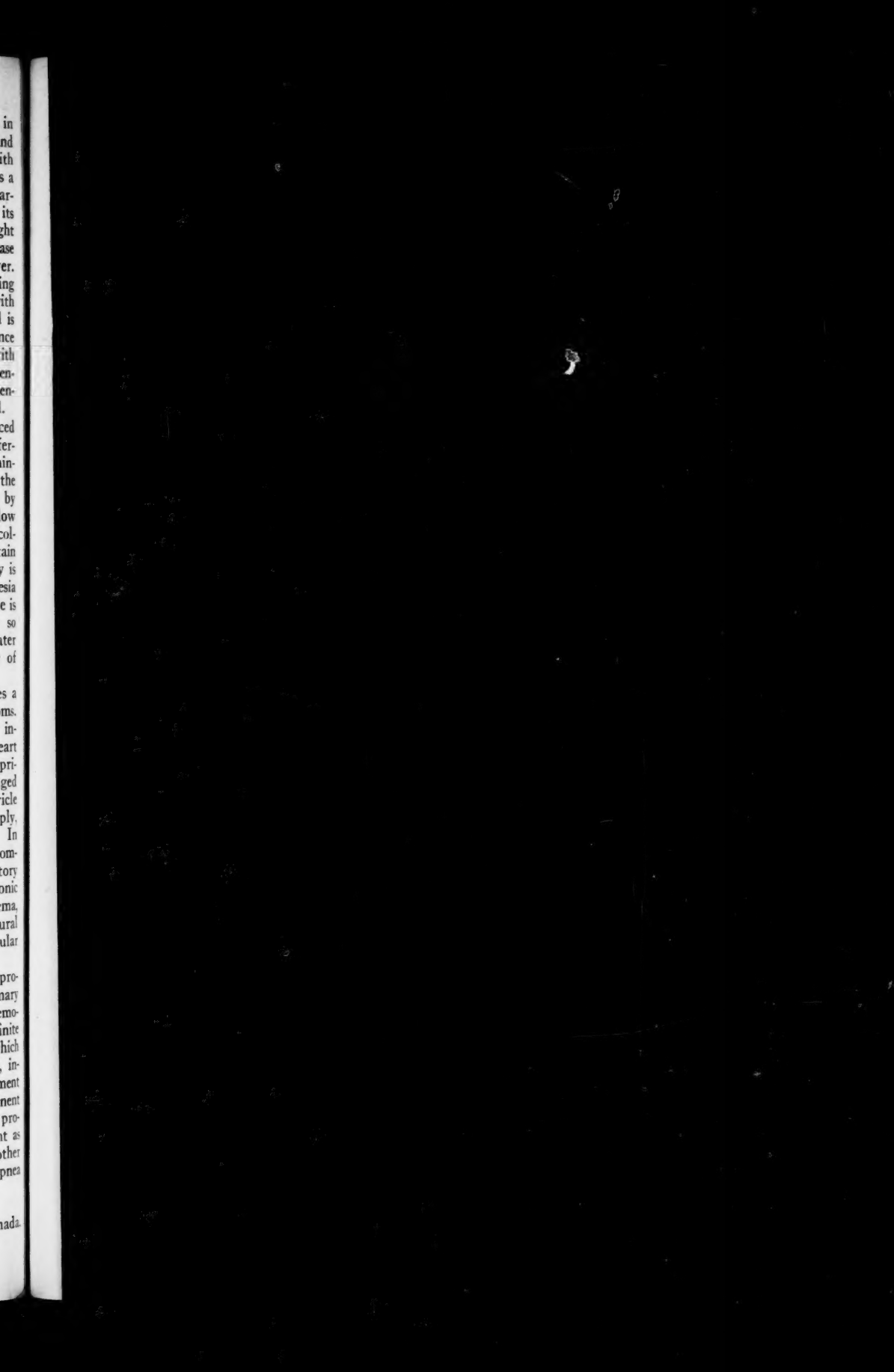
The details of physicochemical changes of the blood and tissue fluids are too numerous and complicated to be considered at present. Loeb has admirably summarized the matter as follows: "Dilatation of the capillaries and veins due to the faulty functioning of the circulation is the principal cause of the increased transudation and the subsequent edema in 'heart failure,' but there may perhaps be in addition an increased filtration pressure responsible for these results. The vascular dilatation is apparently followed by an increased permeability of the capillaries and by a movement of sodium chloride and water in the direction from the blood to the tissues. Retention of sodium chloride plays a rôle also in the case of cardiac edema, although it is a smaller one than in the case of renal edema. In certain stages of cardiac edema a hydremia seems to occur. As to the occurrence of a true plethora, the statements are contradictory. In addition to the increased transudation, there is a delayed absorption and an alteration in the relations determining the exchange of fluid between tissues and blood vessels."

The presence of tissue oxygen want, lactic acid accumulation, decreased pH of the tissues, changes in capillary permeability and numerous other factors, play a variable rôle from case to case, but the fact remains that all of these and edema are due to faulty peripheral circulation and are probably interactive one upon the other. There seems little doubt that the tense brawny edema of extreme heart failure plays an important rôle, through sheer pressure, in still further impairing circulation, thus acting as a vicious circle.

Enlargement of the liver is a fairly early indication of heart failure. There seems but little doubt that this is due in the first instance to the same causes as produce dependent edema. On account of its peculiar position in the circulatory system, early slowing of the blood flow through the capillaries is apt to occur. It is the only organ in the body which receives the vast majority of its blood from a capillary source, namely, the splanchnic circulation. It is to be expected, therefore, that a faulty circulation leading to dilatation of capillaries and veins would produce a conspicuous change in such an organ. In addition to this disturbance of the onward circulation, any increase in vena caval pressure would have a pronounced and early effect through the short hepatic vein. With increasing pulmonary engorgement there follows a steady rise in the pressure in the pulmonary arteries and right ventricle which, laboring in its efforts, promotes increased pressure in the right auricle and great veins, with conspicuous increase in the general venous pressure and also in the liver. But a climax is reached when the tricuspid ring dilates, leading to incompetence of the valves with the result that at each ventricular systole blood is driven back into the great veins and liver, hence the venous and hepatic pulsation synchronous with each heart beat. In acute cases the pain and tenderness over the liver is extreme and acute appendicitis or cholecystitis may be wrongly diagnosed.

Oliguria is an important feature of advanced heart failure, not only on account of the interference with renal function but also as the albuminuria which is frequently present may lead to the erroneous diagnosis of nephritis. It is caused by the slow and diminished volume of blood flow through the kidney. The urine is scanty, high colored, with high specific gravity and may contain albumin, blood and casts. If the specific gravity is low, a primary contracted kidney from hyperpiesia is to be suspected. Maintenance of water balance is one of the principal function of the kidney, so when faulty circulation interferes with this, water retention contributes towards the development of edema.

Heart failure of the *right ventricle* produces a quantitative change in the signs and symptoms. Reference has already been made to tricuspid insufficiency developing as part of general heart failure. The heart failure of hypertension is primarily left ventricular failure, due to prolonged increase in the load placed upon the left ventricle and to progressive decline in the blood supply, through changes in the coronary arterioles. In primary right ventricular failure, a similar combination of events may occur but the circulatory resistance is in the lesser circuit, due to chronic pulmonary fibrosis, such as occurs in emphysema, pneumoconiosis, kyphoscoliosis, extensive pleural fibrosis, and those rare cases of pulmonary vascular disease grouped under Ayerza's disease.

In this type of failure the cyanosis is out of proportion to the dyspnea on account of the primary pulmonary lesion grossly interfering with hemorespiratory function. There is usually a definite polycythemia, due to prolonged anoxemia, which emphasizes the cyanosis. Venous engorgement, increased venous pressure, and hepatic enlargement with or without positive pulsation, are prominent features. The degree of dyspnea is not as pronounced in comparison to the cyanosis in right as in left ventricular failure. Also there is another distinction in that nocturnal paroxysmal dyspnea is comparatively rare.

J. C. Meakins, M.D.
Montreal, Canada.